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Nature, Nurture and the Future of the Analysis of Variance[1]

G.-J. Vreeke

Faculty of Social Sciences, University of Amsterdam, The Netherlands

Key Words

Analysis of variance · Behavior genetics · Cause and variation · Child rearing · Development · Dynamic systems · Interaction and additivity · Nature and nurture · Shared environment

Abstract

Various studies in behavioral genetics have shown that the shared environment does not explain a large part of the variation in the behavior of children. From this finding it is concluded that the family does not have much impact on the development of children. This conclusion (and similar ones) rests on the assumption that findings concerning variation in behaviors can be translated into terms of development. However, the possibility of a translation is not self-evident, as various critics have pointed out. The assumptions on which the method is based and which yields the data of behavior genetics does not match reality, according to critics. The statistical method assumes additivity, while reality is interactive. In this paper it is argued that a translation of behavior genetic data in terms of development requires an interpretative device, which is modelled on the notion of development as a dynamic interactive process.

Behavior genetic studies indicate that the unshared environment – that is, the differences in experiences and events children encounter, and heritability (genetic effects) explain the bulk of the variance in the behavior of children. Surprisingly, the shared environment – that is, the shared experiences of children in a family – explains only a small part of the variance. So when persons share genes this often goes hand in hand with sharing behavior, whereas sharing only experiences hardly ever matches behavioral similarity. In behavioral genetics, specific far-reaching conclusions are drawn from these findings. It is inferred that the family has a limited say in the development of

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G.-J. Vreeke, Faculty of Social Sciences
Department of Education, Universiteit van Amsterdam
Wibautstraat 4, NL–1091 GM Amsterdam (The Netherlands)
Tel. +31 20 525 1242, Fax +31 20 525 1200
E-Mail gertjan@educ.uva.nl

children, that unshared (e.g., accidental and self-induced) experiences together with genetic factors determine the course of development [cf. Jensen, 1997; Rowe, 1994; Scarr, 1992, 1993, 1995; see also Harris, 1998].

The conclusions seem to follow quite naturally. Yet there is an important difference in statements about variation and statements about development. The first type of statement rest on the measured correlation between behavior and their alleged sources. Conversely, a developmental statement is a causal statement regarding the sources of behavior. Behavior geneticists acknowledge that statements concerning variation differ from statements about development. Yet given certain premises and background knowledge, they (seem to) hold that a translation is possible and valid. Authors like Scarr [1992] and Rowe [1994] maintain that the finding of behavior genetics have educational and political consequences. If the shared environment (including parental style, home environment, socioeconomical status) does not explain the difference in cognitive ability and one can conclude from this that parenting does not influence children's development, then one can easily draw the further conclusion that enrichment programs for families are a waste of time and energy. This conclusion rests entirely on the premise that behavior genetics is relevant for questions concerning development.

In this paper, I questions that assumption. More specifically, I shall question whether outcomes of an analysis of variance can be validly translated into terms of development. This paper is not an argument against behavior genetics, far from it. My aim is to question conclusions drawn by behavior geneticists on the base of a specific analysis, the analysis of variance. I think, nevertheless, that the arguments do apply to all correlation-based statistics used in behavior genetics such as multivariate analysis, path analysis, and process orientated techniques that use time series data. However, the arguments do not apply to qualitative forms of research in behavior genetics, such as linkage analyses, nor are they directed at the use of non-linear statistics (although I shall pay some attention to those types of statistics at the end of this paper). Furthermore, even though I question developmental interpretations drawn from data about variance, I shall not be arguing that no translation is possible. My point is rather that there is a gap and that it has to be reckoned with. Behavior geneticists do begin recognising that gap, but they do not take it into account when thinking about development. This paper contains the beginning of a systematic exploration of the meaning and implication of this discrepancy, and thereby aims at the genesis of reflective behavior genetics.

I begin this paper, in the next section, with identifying some of the ways in which behavior geneticists have drawn developmental conclusion from variance analysis. Then I shall review the criticisms levelled against behavior genetics, as far as it relies on the analysis of variance. Can we conclude from these criticisms that developmental conclusions cannot be drawn from those analyses? Following that, I survey the relevant issues necessary for answering this question. Subsequently, I start an exploration as to how data of behavior genetics might be related to development. Finally, the last section lays out an agenda for the future of the analysis of variance.

Developmental Claims in Behavior Genetics

Behavior genetics is traditionally described as the study of the sources of individual differences in a population concerning various traits (such as IQ, personality traits, attitudes, and psychopathologies). The analysis of variance in combination with specific

research designs, especially twin and adoption studies, makes it possible to separately weigh the impact of nature and nurture (i.e., genes and the environment) on behavior. The total variance in a trait is divided in environmental and genetic sources. The environmental sources are further subdivided in a shared and a nonshared component. The genetic sources can also be subdivided in different kinds of genetic effects [for an overview, see Jensen, 1997]. An analysis of variance informs one on the differential effects of genes and the environment in the behaviors of persons *in a sample*. The validity of an analysis of variance is thus restricted to a given population. Critics also point out that type of measurement and kinds of genetics comparison (e.g., parent-child, siblings) are known to influence heritability estimates [cf. Hoffman, 1991].

Behavior geneticists often oppose the equalling of behavior genetics with the analysis of variance. They use other statistical techniques, are engaged in qualitative oriented research and claim to possess a material theory that is basic to their use of statistics (which is – roughly put – a combination of evolutionary theory and Mendel's laws). Though all this is correct, research in behavior genetics on complex traits like personality, intellectual abilities, or attitudes is predominantly a quantitative affair. And in this type of research the analysis of variance and its relatives (all correlation based statistics) play a central role. Our description of behavior genetics is thus by no means complete, but it does cover an important part of the research. Now, which developmental claims are commonly derived from analyses of variance?

A brief inspection of the nature of a development claim is in order. A developmental claim is a claim concerning the mechanisms of development [Gottlieb, 1995]. A 'mechanism' is any describable causal process [Brandon, 1990, p. 185]. So a developmental claim can be understood as a judgement in which a cause of a certain behavioral outcome is described. A cause can be conceived as an agent (or a collection of agents) operating in a given context, which helps to produce an outcome. Help can be given in various ways. So there are various ways in which an agent can aid the realisation of a particular outcome. It can completely produce its occurrence, facilitate it, remove a barrier to its occurrence, it can 'not prevent and not avoid it' (e.g., provide opportunities), and so on [cf. Nozick, 1993, p. 61]. A developmental claim can apply to individuals or to populations, it can (thus) be probabilistic or definitive. Making a developmental claim, then, amounts to identifying the (collection of) causes (agents of change) in a given context, which aids a behavioral outcome in one way or another.

A first way in which variation is related to development is by translating the percentage a source of variations explains in terms of the causal effect of that source. So, if the heritability of IQ in a population is 80%, one can say of an individual in that population who has an IQ of 120 that 80% of the deviation of the mean is probably due to genetic factors [cf. Plomin, De Fries, McClearn, and Rutter, 1997]. Thus genetic factors are considered to be highly responsible for differences in IQ. This, then, is generally understood as indicating that genetic factors have a large causal impact on intellectual abilities. However, what kind of causal effect is present, is not spelled out.

Studies in behavior genetics, which use the analysis of variance, as a rule offer this type of developmental conclusion. The models or causal paths, which are offered in those studies, witness this state of affairs. It is generally assumed that the assessed correlations in an analysis of variance are manifestations of underlying causal patterns. Offering developmental interpretations of an analysis of variance is thus as standard practice in behavior genetics. It is thus assumed that the material base of behavior genetics is solid enough to allow for those developmental conclusions.

However, even if that is the case, inferences about development do not follow automatically. If there is no information available about the sample (e.g., how it relates to the general population), the heritability estimate is not very informative with regard to the causal effects of genes on behavioral outcomes. If a sample consists of successful college students with similar backgrounds, we could find that genes make all the differences in cognitive abilities. Yet if the same individuals were part of a much larger sample, in which persons with widely different backgrounds were assessed, perhaps the environment would have explained the major part of the variance in IQ. Thus a different causal explanation would follow. Interpretations of analysis of variance in terms of development need the aid of background knowledge, especially information about the relation between the samples and the general population. One can obtain that information by using measures on different types of samples, or by estimating the extent to which one's sample differs from the general population.

In his book *The limits of family influences* [1994], Rowe refers to a wide variety of data concerning the sources of individual differences. He points out that data related to personality (introversion, extraversion and so on), IQ – measures, and measures of attitudes, all show a similar pattern. With some minor exceptions, these behaviors have a large genetic component, as well as a large unshared environmental component. The shared environment explains only a small percentage of the variance related to those traits. According to Rowe, it follows that the shared environment has a minor influence on the development of children, whereas genes and the unshared environment have a major impact on development. On the effect of the shared environment, Rowe says, '... for many personality and intellectual traits, variations in shared family environment have little influence on trait development. ... The effects of shared family environments on children's developmental outcomes are limited' (p. 53). It is clear, then, that Rowe relies on the idea that percentage of explained variance is an indication of the causal strength of a variable in development. If the shared environment again and again does not explain the variance in types of behavior in a population, one may conclude that it does not have a major effect on children's development in general. Thus, no causal process can be conceived as (effectively) present.

A second way in which variation is related to development is by predicting behavioral outcomes on the basis of heritability estimates and/or other components of variation. This kind of relationship between variation and development has direct practical relevance. It helps assessing the chance that children of parents with a certain psychopathology (eating disorders, mood disorders, and schizophrenia) are in danger of developing it too. If an analysis of variance indicates that depression has a heritability of 20% in a population, and a child has one parent who suffers from this major mood disorder, one can estimate the chances of the child developing the disorder. This estimate is expressed in terms of a risk percentage [cf. Plomin et al., 1997].

This type of inference from variation to developmental conclusion is useful, but not very precise. Furthermore, it is assumed that the environment in which the child develops will not radically differ from the environments in which the heritability estimate was established.

Again the inference relies on the assumption that a percentage of variation explained by a variable informs us about the relative causal power to produce a certain behavioral outcome.

This is also apparent in the views of behavior geneticists on the impact of deliberate interventions designed to further children's development. An intervention is gener-

ally considered a deliberate change in the shared environment. Scarr [1992] takes up the fact that the shared environment does not account for much of the variation in personality traits, IQ, and the like, as the primary source of evidence that deliberate interventions will not aid development to a considerable degree. Her argument is that when the shared environment in general explains a small percentage of the observed variance, the chance that changes in the shared environment will have a great impact on the trait will be small too. This reasoning is also evident in Jensen's [1969] well-known article in the *Harvard Educational Review*: 'Can we boost IQ and scholastic achievement?' [cf. Bereiter, 1987 for an informative analysis]. The amount of variation a variable explains is thus taken to describe the probable effect of (environmental) changes. In other terms: When a variable explains a certain amount of variance, this is taken as an indication of the margins of change by the manipulation of that variable. If, for instance, differences due to the shared environment explain 1% of the variance in behavior, changes in the shared environment will not make much difference in general. If the shared environment explains a great deal of the differences in behavior, changing the rearing conditions thus could make a lot of difference. As Rowe [1994] states: 'A phenotype might be changed by altering the rearing conditions. The greater the shared rearing estimate, the more change can be expected to follow from changing rearing conditions' (p. 33).

Scarr [1992, 1993] has tried to include population characteristics in her theorising in a novel way. Scarr explicity explores the idea that an analysis of variance has to be understood against a background of what is shared by persons in a population. These population effects (which include biological and environmental features), when explicitly taken into account, do indeed alter developmental conclusions. Basic to Scarr's view is the finding of behavior genetics that genetic similarity correlates with various types of behavioral similarity. Yet Scarr acknowledges that development depends on environmental sources and that many of these sources do not differ from individual to individual. Most children in the Western world grow up in families or family-like figurations, receive stimulation and have contact with peers, go to schools, and so on. This then brings Scarr to conclude that in normal circumstances, genes (relying on those circumstances) produce behavioral outcomes which largely reflect their genetic potential. Extra stimulation (etc.) will not have a positive effect on the social and intellectual development of children, if these children are already within what she calls 'the normal range'. That is, if they have been able to profit from the environmental sources that facilitate development (e.g., growing up in a family with (normal) parents or persons, who act like parents, receive at least average stimulation, and so on). When children are outside the 'normal range' they can and will probably profit from deliberate interventions, if these interventions provide these children with the range of experience apt to their (normal) genetic potentials.

Criticism on the Additivity Assumption

The analysis of variance rests on the assumption that the variables responsible for behavior are separate and can form additional explanations of behavior. If this assumption reflects the actual relation between genes and environment, a translation of variance in terms of development is possible – if one succeeds in taking into account population effects. The additivity assumption, however, is subject to criticism. Critics have argued that the additivity assumption, on which the analysis of variance is based, does not fit actual developmental processes. Lewontin [1974] points out that the actual

causes of behavioral development (environmental and genetic) do not add up in most cases. Experimental animal research shows that interaction between genotype and the environment occurs often. And if genes and the environment interact, it is not possible to separately weigh the effect of one of those factors: they depend on each other. There is no reason to expect that humans are different in this respect. An analysis of variance ignores those effects, so cannot provide a true account of the causes of behavior. Wahlsten [1990] has shown that the analysis of variance, though it can in principle detect interaction, is too insensitive to do so. As a rule, then, variables are considered as separate, that is, as adding separately to a behavioral outcome. To a large extent this is an artefact of the method used (later in this section I shall elaborate on this criticism). Gottlieb [1995] observes that molecular genetics supports a model of the relation between genes and the environment as 'interactive all the way down'. Genes should no longer be seen as encompassing a blueprint for development, but rather as coactors in a multileveled bi-directional system. Wade [1992] observes: 'Genes interact within and between loci in the development of most phenotypes, and the existence of these interactions prohibits a simple interpretation of the observed additive genetic variance in terms of additive gene action' (p. 150).

The core of the critique of behavior genetics, as far as it relies on the analysis of variance, is thus that it conceptualises the relation between genes and the environment as (mainly) additive, whereas in fact development is interactive. Because of this mismatch no (simple) translation from an analysis of variance to development is valid, according to critics. I will spell out the arguments underlying this criticism more fully. To do so we should first look closer at the differences between addition and interaction.

Addition and Interaction

Additivity and interaction can be understood best from a developmental perspective, that is, as saying something about the way variables are causally connected to each other. From this perspective, then, it becomes possible to look at research, which in turn provides the clues for a developmental interpretation.

On a broad understanding, interaction means that when two (or more) factors are needed to produce a result, there is interaction. Interaction thus means something like 'necessary to produce an outcome'. On this understanding development is interactive by definition, for it needs the cooperation of various organic and non-organic elements. In this sense behavior genetics deals with interaction.

However, in order to differentiate interaction from additivity, we must look for a more narrow interpretation of interaction. We can do so by applying this notion (and that of additivity) to the nature of the relationship between variables (in this case: genes and environment) and developmental outcomes (behavior). Interaction pertains to a relation between variables in which the effect of one variable on a developmental outcome depends on the value of other variables, whereas additivity refers to an additive relation between various variables.

When variables have an additional impact on behavior, they are separately contributing to a behavioral outcome; when the variables interact they are interdependent, implying that the behavior is to be seen as the result of the interplay between various sources. If the relationship between variables is additive, this also implies that the variables *correlate* with behavior [cf. McCall, 1991, p. 144]. In other words, the presence of a

certain type of behavior goes hand in hand with a certain amount of genetic and environmental similarity. If interaction effects are the whole story, no correlation is found. In that case it is impossible to trace down the impact of genes and the environment separately.

Now let us look at research. Suppose we have no information about the way in which various sources produce behavior. However, we find that genetic similarity correlates with behavioral similarity, and – though to a lesser degree – environmental similarity also correlates with behavioral similarity. Accordingly, the behavior of persons in a population can be explained in terms of additivity, e.g., 80% of the variation is due to genetic effects, 20% stems from environmental sources. Because of the fact that actual additional effects can induce correlation, one might assume that the additivity assumption holds true. And this implies that one might infer – in this case – that genes have a larger causal effect on behavior in comparison to that of the environment.

When no correlations are found in one's data, this can mean various things in terms of development: that the effect is mediated by a third (not measured) variable, that the outcome depends on the specific values of the various variables, that the phenomenon has a chaotic nature, and so on. All those effects can in principle be labelled as interactive. Interaction then is assumed when the effect of a variable on an outcome is *nonlinear*.

In the research literature, however, we find that the notion of interaction is applied to *specific* nonlinear relationships between variables and outcomes. Plomin et al. [1997] speak about interaction when a specific genotype is sensitive to a particular environmental condition. (This would be the case if 'intelligent' genes produced intelligent behavior only in highly enriched environments.) Loehlin [1992] defines interactions as the process in which two variables with various values produce the same outcomes (e.g., when high intelligence results from enrichment of persons with poor genetic potentials and also from a lack of stimulation of persons with rich potentials). However, there is no good reason to restrict the notion of interaction to these specific effects. We can label all nonlinear effects of genotype and environment on a developmental outcome as interactive.

So finding correlation between genetic similarity and outcome data are likely to be interpreted as indicating that genes and environmental influences on behavior have an independent impact on behavior. And because of the fact that, in general, interaction effect is not found, it is assumed that the additivity assumption holds true. The developmental conclusions we discussed in the previous paragraph, indeed, all rely on a conceptualisation of genes and the environment in terms of additivity, as 'separately contributing to behavior'.

Material and Statistical Assumptions

Keeping this elucidation of additivity and interaction in mind, let us take a closer look at the criticisms levelled against behavior genetics reliance on additivity.

Wahlsten [1990] points out that interpreting outcome data in terms of processes which led to those outcomes requires that the statistical assumptions concerning the relation between variables match material assumption with regard to relation between genes and the environment. As stated, Wahlsten argues that the ANOVA (analysis of variance) procedure is too insensitive to find interaction. In order to show this, Wahlsten computes the chances that interactions are found in a data set in which the relationships between variables are known to be multiplicative (interactive). He finds that main (that is, additive) effects are easily shown, but that the chances that the relationship between the variables is modelled in interaction terms is considerably lower. ANOVAs

then are not very powerful when it comes to finding interactions (pp. 114–116). Wahlsten considers two reasons why this might be so. One reason is that the interactions that (might) occur on the individual level get lost in assessing the overall relationship between the variables. To that end, mean values are computed, which harmonises the different ways different groups with the same genotype react to various environmental conditions (p. 116). Another reason Wahlsten considers this is that the interactions that occur do not follow a consistent pattern, and thus fail to become significant (p. 113).

Additive Reality

Wahlsten's paper has received various comments. Some authors think that Wahlsten's criticism is unwarranted [e.g., Plomin, 1990]. They reject the view that reality is interactive. According to them, there is evidence for the view that reality is (mainly) additive. Quantitative genetics is not just a statistical device. It is also a theory that is grounded in genetic theory [Plomin, 1986].

Mendel's laws and evolutionary theory are called to the stand to offer support for the additivity assumption. Mendel's laws are derived from studying single genes in controlled environments. And although there are exceptions to those laws, they do offer a solid base to generalise from. Mendel's laws can be applied to traits, which are influenced by multiple genes: cognitive ability, personality characteristics, and so on. The multiple genes that play a part in complex traits such as these consist of various single genes, which are inherited according to Mendel's laws. The effect of multiple genes on behavior, furthermore, can be taken as generalised effects of single genes. In turn, the impact of the environment can be treated as an effect similar to that of a genetic one. So if you have a population in which you can estimate the amount of genetic relatedness (a population of twins, siblings, etc.), you can also – on the basis of the single case model – estimate the various genetic and environmental components of variance in that population [cf. Plomin et al., 1997]. In short, behavioral genetics relies on a small set of material assumptions (Mendel's laws) from which it is possible to generalise.

However, these assumptions do not buy the additivity hypothesis. Mendel's laws mention, apart from additive relations, interactive effects (e.g., epistase) between genes. So there is no reason to assume that the relationship between genetic factors and the environment should be modelled mainly on additivity. The truth of the matter might be that in generalising, interactive effects could be ignored, which is different from saying that behavior genetics is based upon a corresponding material assumption.

Yet, there seem to be substantial reasons why the effect of the environment can be added to that of genes. In response to Wahlsten, Cheverud [1990] points out that heredity analysis is developed to provide the missing link between Mendel's laws and evolution. Crow [1990], McCall [1991], and Goodnight [1990] use this connection to argue that development is essentially additive. The reason is that measured traits or phenotypes are – that is: can only be seen as – a product of evolution. According to Crow [1990] we can see adapting to an environment as a process in which an increment of a function (such as a capability) is realised: organisms gain in length, become more intelligent, develop better protective skills, etc. Adaptation, therefore, can be understood in terms of additivity, genes *plus* the environment result in the increment of the function or capability. Thus, gains in intelligence, length, or whatever, can only be understood as resulting from the additive effects of genetic potential and the environment. In short,

nature favors addition over interaction because the first is more adaptive [cf. McCall, 1991, p. 145].

These arguments establish a connection between adaptation and additive effects in a population, but it is far from clear that they can be generalised to individual development. The additive effect of genes and the environment on behavior is not due to a material inbuilt characteristic of genes or the environment, but is more likely the result of ignoring the steps that lead from genes to behavior and of using broad phenotypic categories to conceptualise developmental outcomes. It is thus an assumption mostly based on statistical reasoning, which does not amount to grounding behavior genetics in a more basic material theory. In short, no conclusive material reason for adopting the additivity assumption is offered.

Furthermore, evolutionary theory seems by itself unsuitable for treating the variables at stake as separate. The evolutionary explanation itself focuses on results (e.g., the fact that organisms adapt to changes in the environment). These results can be meaningfully interpreted as an improvement of adaptations (which by the same token can be considered an increment in additive effect), but this does not imply that these results stem from the *actual* additivity of genes and enviroment. The process by which the effect is realised can still be interactive.

Additivity as a Model

Although some authors seem to hold the view that reality is (mainly) additive, it is not always clear whether they hold that view because they think it offers a suitable model, or because they really think that development is actually the result of genes and the environment adding separately to an effect. My guess is that the view that genes and the environment are additive forces is not held in earnest by anyone. What makes the view that 'reality is additive' so problematic is that it must ignore evidence from molecular biology and experimental animal research [cf. Gottlieb, 1995]. The process by which genotypes get transformed in phenotypes is (if we may rely on those findings) complex, dynamic, and nonlinear, which clearly contradicts a material view that holds that the relation between genes and the environment is mainly additive [cf. Wahlsten & Gottlieb, 1997; see also Oyama, 1985].

This fact, and absence of material support for the additivity thesis, implies that one cannot claim that the analysis of variance matches the action of genes in an organism in relation to an environment. As far as I can see, no one will explicitly adhere to the additivity thesis in this form [cf. Turkheimer, Goldsmith & Gottesman, 1995; Scarr, 1995; Burgess & Molenaar, 1995; Dolan & Molenaar, 1995]. One rather sticks to the thesis because it offers a suitable model for studying the sources of individual differences in a population, even though it does not match reality perfectly. So, from the conclusion that reality is interactive, one cannot infer that the analysis of variance is invalid, *because* it does not match with material assumptions. One acknowledges that there is a mismatch, but seems to think that this is inevitable – given our state of knowledge. Yet the results of an analysis of variance are (given a careful use) ultimately valuable [cf. Scarr, 1995].

In view of this, however, it is surprising that the authors who express this view do not inform us about the precise ways in which variation and development can be linked. At most we are informed that care is needed in these matters [cf. Turkheimer et al., 1995; Scarr, 1995].

Relating Variance and Development

We are left with the question of what analyses of variance can tell about development. And thus with the question of how we should evaluate the inferences about development that various authors have drawn from behavior genetic studies.

If results of the analysis of variance can be translated meaningfully in terms of the interactions that 'really' take place, variance analysis would not be at odds per se with an interactive reality. Indeed, this point of view seems the dominant strategy of researchers who view development as an interactive 'all the way down', but still want to make use of the tools of quantitative genetics. These techniques are considered of comparable power compared to experimental designs, designs which cannot be used anyway with complex behavioral phenotypes like intelligence and personality traits [Scarr, 1995].

We now arrive at a crucial problem, which I think has not received the attention it deserves in the literature. If you hold that reality is interactive, but use methods that presuppose the separateness of genes and the environment, you are stuck with a problem of interpretation. At least if you want to say something about development, about what has caused a behavioral phenotype to occur. What can your data tell?

If you hold that a translation can be made, you must presume that when one conceives variables as separate, this is a meaningful abstraction from interactive reality. This in turn implies two things. One is that you must have an idea how to model the material process with regard to development. Such modelling requires an interactive theory, for this after all is how reality is to be conceived. Thus a model is needed that traces a behavioral outcome to interdependent sources on various levels. Of course such a model can hardly be descriptive, for in most cases we know too little about the precise mechanisms. Yet it should have sufficient levels and variables to make a realistic mapping possible. But this is not enough. What is needed in the second place are guidelines as to how one should map the outcome of an analysis of variance in terms of the process model. In order to accomplish this, one needs an understanding in what way an interactive (dynamic) reality can be mapped as dominantly additive.

Some clues how this is possible are provided by Crow [1990] and can also be abstracted from Wahlsten's [1990] analysis.

Crow [1990] maintains that the analysis of variance is sensible and suitable at the level of the *results* of the developmental process. What happens at a basic material level, according to Crow, can be viewed from the point of view of effects, as additive causes. Every molecule in a gas behaves in its own peculiar ways and has unique encounters with neighboring molecules. When we compress gas to turn it into a fluid, the behavior of a single molecule – as a result of this environmental change – is unpredictable. Yet the effect on the totality of the gas molecules can be predicted perfectly. Crow's suggestion is interesting, but has the worrying implication that the level of analysis codetermines how development is to be understood. This may result in a multi-leveled view of development, where on every level a different story is told, incompatible with the stories told at other levels. Surely, we want an explanation of the behavior of a gas molecule in a particular environment, compatible with an explanation of how gas is compressed to a fluid.

However, what we learn from Crow is that the perspective one takes makes a difference with regard to the (in)determinancy one finds in a system. Looking at the molecular level, we find chaos, indeterminacy that is interactions; however the effect of all those indeterminate actions is grossly similar when various cases are observed: the gas

turns into a fluid. This example can be extended by many others. The process of motor development is another example. Motor development is the result of the interplay of a huge variety of elements [cf. Thelen & Ulrich, 1991]. The various interactions that occur during the learning (developmental) process are unique to an individual; nevertheless, the result of all those interactions is largely similar: most children learn to walk. Likewise, from a genetic perspective it is all interactions we watch, whereas from the population perspective we only see the product. Observing similarities in effects, we naturally assume a similarity in process, which then drives us in the hand of a comprehensive additivity model.

Wahlsten [1990] concludes his paper by noticing that choosing larger samples increases the chance of finding significant interactions. Earlier he also noticed that increasing the amount of strains and environments had a similar effect. This, then, amounts to the postulation of a surprisingly simple rule: The more variables one distinguishes in a study, the more likely one will find interactions. Having two sources of variation (genes and the environment) implies that underlying interactions will fail to get noticed: they do not show up as differences in behavior, are tied to specific genotypes (e.g., do not generalise), etc. Authors who gave testimony of the problems in behavior genetics with finding interaction often blame this on the use of estimations of the environment. When the environment is measured you gain an extra variable and can actually measure how it reacts (interacts!) with various genotypes [cf. Eaves, Eysenck & Martin, 1989].

The lesson to be drawn from this is that the difference between experiential research (including molecular biology) and research in behavioral genetics is one of focus (levels) as well as one of *numbers*, e.g., the type and amount of variables included in a study. If one studies outcomes, the results are 'how much' data, and if there are two variables distinguished, one is likely to miss various underlying interactions. Knowing this can help to arrive at (more) suitable interpretations of behavior genetic research.

Reinterpreting Developmental Conclusion of Behavior Genetics

With this analysis in mind, I want to take a short look at some of the examples discussed earlier. Scarr [1992] holds, as we saw, that an average expectable environment makes normal development possible, while the shared family environment hardly effects children's development. So Scarr argues that the effects of 'average expectable' environments are different in type from those of the shared environment, thus assuming that different kinds of causal relationships hold in these situations (a facilitating versus a 'not providing, not preventing' relationship). Average expectable environments facilitate development because they provide the necessary ingredients for (normal) development, whereas specific family influences in general do not show (alike) effects because children react differently – according to their individual genetic potentials – to the stimuli; those influences then do not provide, yet also do not prevent, development.

Baumrind [1993] and Jackson [1993] have pointed to various counterexamples with regard to Scarr's [1992] view that extra stimulation does not promote development. Scarr's [1993] response to these examples is that these are nonsignificant exceptions, which is plausible. However, from an interactionist point of view, there does not have to be a contradiction between these findings. Experimental social research in general does not control for genetic factors, but does compare the effects of various environ-

mental influences on behavior. Doing so enlarges the change of tracing effective input, that is, input which fits the needs and potentials of a specific group of children. Still it is highly possible that those environmental influences would not show up in a behavior genetic study. This can be explained in terms of behavioral genetic assumptions. Behavior geneticists hold that genes and experiences connect – genes drive experiences [cf. Scarr, 1992; Bouchard, Lykken, Tellegen & McGue, 1996]. This might preclude finding significant effects of the shared environment. A common environmental effect does not show up, because persons with different genotypes tend to react differently to the same stimulus. And because research designs in behavior genetics in general do not relate specific environmental input to specific genotypes, the effect of extra stimulation on the development of a specific group of children will not show.

As a consequence of their views on development, interactionists and behavior geneticists also differ with regard to the way in which they conceptualise educational and family influences. Behavior geneticists will emphasise main effects of the shared environment. But from an interactionist's point of view, the fact that the shared family environment does not explain a large part of the variation in behavior, does not show that the family upbringing has a limited effect on development. From this point of view, one does not expect that there is a linear relationship between what parents do and how their children develop. So the view that family influences should show up as a large percentage of the shared environment is not part and parcel of the interactionist point of view.

Furthermore, the view that the shared-environment estimate indicates the probability of change when rearing conditions are altered is misleading. It is misleading because this view applies paradigmatically to cases where one is ignorant about what aids development. But this does not imply anything about the (actual) limits of family influences (or other environmental effects). Take growth for example. The shared environment does not explain differences in average height in a normal population. An ad random change of environmental influences, and probably even one that is more to the point (altering diets for instance), will not affect average heights. However, one can influence growth considerably if the right measures are taken. In this case, one can rely on the fact that growth is partly dependent on hormonal factors. Treating children who have growth problems with hormones might help them to reach more normal heights. Even if the variance in height cannot be explained by the shared environment, an environmental – or rather a 'nongenetic' change – can affect development considerably. To be sure, those interventions do not always have to include a direct manipulation of biological variables. There is no reason to assume that the stimulation of learning habits, tailored to individual needs, would not aid the growth of intellectual abilities. Of course, one may be pessimistic about stimulation programs, holding that positive effects thereof are short-lived or mediated by unassessed genetic factors. However, it cannot be deduced from percentages of explained variation that an intervention cannot be successful. An interactive logic predicts that it is a matter of finding the right key to the right lock, the environment that fits an individual genotype [cf. Baumrind, 1993].

Non-Linear Statistics and Behavior Genetics

I want to conclude this review by paying attention to the view expressed by Burgess and Molenaar [1995]. They agree with Gottlieb [1995] that an interactive (dynamic) developmental system view accords best with the material relationship between genes

and the environment. Consequently, they argue that 'the developmental "distance" between initial genotype and realised behavioral and cognitive phenotypes is large – a distance has to be bridged by a network of epigenetic systems whose non-linear dynamics destroy genetically induced correlations' (p. 160). Still, they hold that behavior genetic studies provide valid information concerning the causes of development: 'We observe in behavior genetic investigations that these phenotypes can show substantial genetically induced correlation between relatives' (ibid.). As these finding conflict, Burgess and Molenaar observe the emergence of a paradox, the solution of which is unclear. If our analysis is correct, there should not be a problem with the resolution. The observed correlations cannot readily be interpreted as information about development; in this sense they are partly an artefact of the methods used, which from the onset presuppose the separateness of the variables. So, using methods which presuppose the interdependence of various variables could offer a solution.

In other papers Molenaar and Boomsma [1987] have proposed a nonlinear statistic in order to document interaction. Making use of these types of methods, then, could help close the gap between statistical method – using these new techniques – and an interactive developmental system model. The future of behavior genetics, as far as its quantitative components are concerned, perhaps lies in becoming familiar with those techniques. However, the gap is still there. The database of these alternative forms of analysis are still outcome data (e.g., behaviors), although those alternative forms of analysis require more variables (which supports the idea that interactions are easier brought to light when more variables are selected). But these variables do not cover all the levels of operation an actual dynamic system has, so although using non-linear forms of statistical analysis may be part of the solution, it cannot be the whole story.

The Future of the Analysis of Variance

An analysis of variance offers insight in development in terms of probabilities, but these statements cannot simply be translated in terms of the impact of genes and the environments on (individual) development. An analysis of variance abstracts from (actual) interaction effects and thus cannot offer an accurate picture of development.

In this paper, I have explored two ways of getting ahead, which in my mind should direct the future of the analysis of variance. One is that of learning the process by which an analysis of variance transforms interactive reality into separate effects. Knowing this process enables one to develop interpretative devices that allow for more sensible developmental conclusions. Second, one can aim to model one's research method on the assumption of an interactive reality. Doing so bridges the gap between statistical and material assumptions and thus allows for more accurate developmental conclusions. However, this route can only be followed a short while. I do not think that it is possible to find a match between interactive reality and statistical devices completely. So even if the latter route is followed, one still needs interpretation to arrive at developmental conclusions.

Behavior geneticists, then, should acknowledge that an analysis of variance is a statistical method that does not fit reality and should be judged against the background of the best material model we have of development, which is one of dynamics and interactions. This discussion should proceed discussions about the political or educational impact of the finding of behavior genetics.

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